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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, CANCERLIT, BIOTECHDS' ENTERED AT 14:53:09 ON 04 JUN 2003

L1	3099	S 100K OR NUCLEOTID? 9###
L2	109595	S ADENOVIR?
L3	246	S L2 AND L1
L4	82	DUP REM L3 (164 DUPLICATES REMOVED)
L5	2315221	S DELE? OR REMOV?
L6	632978	S DEFICIENT OR LACKING
L7	2890079	S L6 OR L5
L8	16	S L7 AND L4

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Search: L4

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DATE: Wednesday, June 04, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L4	L3 with l2	22	L4
L3	adenovi$	22259	L3
L2	100K or nucleotide 9??? or nucleotides 9???	6045	L2
L1	100K or nucleotide 9???	6045	L1

END OF SEARCH HISTORY

L8 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2003 ACS
AN 2000:161478 CAPLUS
DN 132:204060
TI **Adenoviruses deleted** in the IVa2, **100K** and/or preterminal protein sequences
IN Amalfitano, Andrea; Chen, Yuan Tsong; Hu, Huimin
PA Duke University, USA
SO PCT Int. Appl., 156 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000012740	A2	20000309	WO 1999-US19540	19990827
	WO 2000012740	A3	20001123		
	W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, CZ, DE, DE, DK, DK, EE, EE, ES, FI, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2340276	AA	20000309	CA 1999-2340276	19990827
	AU 9956942	A1	20000321	AU 1999-56942	19990827
	EP 1108049	A2	20010620	EP 1999-943952	19990827
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	US 6328958	B1	20011211	US 1999-384749	19990827
	JP 2002528056	T2	20020903	JP 2000-567725	19990827
PRAI	US 1998-145742P	P	19980828		
	WO 1999-US19540	W	19990827		

AB The present invention provides **deleted adenovirus** vectors. The inventive **adenovirus** vectors carry one or more **deletions** in the IVa2, **100K**, polymerase and/or preterminal protein sequences of the **adenovirus** genome. In the human **adenovirus** serotype 5 genomes, such **deletions** are at nucleotide positions 4830-5766, 24,990-25,687, and/or 7274-7991. The **adenoviruses** may addnl. contain other **deletions**, mutations or other modifications as well. In particular preferred embodiments, the **adenovirus** genome is multiply **deleted**, i.e., carries 2 or more **deletions** therein. The **deleted adenoviruses** of the invention are "propagation-defective" in that the virus cannot replicate and produce new virions in the absence of complementing function(s). Preferred **adenovirus** vectors of the invention carry a heterologous nucleotide sequence encoding a protein or peptide assocd. with a metabolic disorder, more preferably a protein or peptide assocd. with a lysosomal or glycogen storage disease, most preferably, a lysosomal acid .alpha.-glucosidase. The **deleted adenovirus** vectors advantageously have an increased carrying capacity for heterologous nucleotide sequences, demonstrate lower levels of viral protein expression, induce fewer host immune responses, and/or exhibit increased stability and prolonged transgene expression when introduced into target cells.

L8 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2003 ACS
AN 2002:946147 CAPLUS
DN 138:34131
TI Helper-virus independent replicating **adenovirus** vectors with **100K** or E1b gene **deletion** for gene therapy
IN Amalfitano, Andrea; Hodges, Bradley L.
PA Duke University, USA; Koeberl, Dwight D.
SO PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002098466	A1	20021212	WO 2002-US17070	20020531

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-295914P P 20010604
AB The present invention provides replicating [**100K**-] **adenovirus** vectors that have an impairment in **100K** activity. In particular preferred embodiments, the impairment is the result of a **deletion** in the **100K** coding region of the **adenovirus** vector genome. It is further preferred that the **adenovirus** produces the E1 gene products. In an alternate embodiment, the **adenovirus** produces the E1a gene products, but has an impairment in the E1b coding region, such that replication of the virus is limited to p53- cells. Also described are methods of making and administering the inventive **adenovirus** vectors to a cell or to a subject. Further provided is use of the inventive [**100K**-] Ad vectors as a helper virus for the prodn. of vector stocks of adeno-assocd. virus.

L8 ANSWER 7 OF 16 MEDLINE
AN 83303837 MEDLINE
DN 83303837 PubMed ID: 6612996
TI Analysis of Ad5 hexon and **100K** ts mutants using conformation-specific monoclonal antibodies.
AU Cepko C L; Sharp P A
NC NIH-P01-CA14051 (NCI)
P01-CA26717 (NCI)
SO VIROLOGY, (1983 Aug) 129 (1) 137-54.
Journal code: 0110674. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198310
ED Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831008

AB **Adenovirus** type 5 ts mutants **deficient** in hexon metabolism were investigated using conformation-specific monoclonal antibodies directed against hexon capsomeres and the viral **100K** protein. The ts mutants map either in the hexon structural gene or in the gene encoding the **100K** protein, a major, late nonstructural protein. All of the mutants examined (ts1, ts2, ts3, ts4, ts17, and ts20 of J. F. Williams, M. Gharpure, S. Ustacelebi, and S. McDonald (1971). J. Gen. Virol. 11, 95-101) were unable to produce the capsomeric form of hexon (a trimer of three hexon monomers) at the nonpermissive temperature. However, all of the mutants retained the ability to produce a complex of **100K** and hexon which has been demonstrated to play a major role in the assembly of hexon trimers. The mutants accumulated nontrimerized hexon in this ts complex in the perinuclear region of the cell. Several of the mutants (ts1, ts2, ts3) were found to successfully assemble hexon synthesized at the nonpermissive temperature upon shift down to the permissive temperature, even in the presence of a protein synthesis inhibitor. The mutant, ts2, which maps in the hexon structural gene, was found to be dependent on protein synthesis for transport of hexon trimers into the nucleus during temperature shift down, while the **100K** ts mutants, ts1 and ts3, were independent of protein synthesis for both hexon assembly and transport.

L8 ANSWER 1 OF 16 MEDLINE
AN 2001320301 MEDLINE
DN 21286721 PubMed ID: 11390592
TI **Adenovirus** vectors with the **100K** gene **deleted** and their potential for multiple gene therapy applications.
AU Hodges B L; Evans H K; Everett R S; Ding E Y; Serra D; Amalfitano A
CS Department of Pediatrics, Division of Medical Genetics, Duke University Medical Center, Durham, NC 27710, USA.
NC DK52925 (NIDDK)
SO JOURNAL OF VIROLOGY, (2001 Jul) 75 (13) 5913-20.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200106
ED Entered STN: 20010702
Last Updated on STN: 20010702
Entered Medline: 20010628
AB The **100K** protein has a number of critical roles vital for successful completion of the late phases of the **adenovirus** (Ad) life cycle. We hypothesized that the introduction of **deletions** within the **100K** gene would allow for the production of a series of new classes of Ad vector, including one that is replication competent but blocked in the ability to carry out many late-phase Ad functions. Such a vector would have potential for several gene therapy applications, based upon its ability to increase the copy number of the transgene encoded by the vector (via genome replication) while decreasing the side effects associated with Ad late gene expression. To efficiently produce **100K-deleted** Ad ([**100K**-]Ad) vectors, an E1- and **100K**-complementing cell line (K-16) was successfully isolated. Transfection of an [E1-,**100K**-]Ad vector genome into the K-16 cells readily yielded high titers of the vector. After infection of noncomplementing cells, we demonstrated that [**100K**-]Ad vectors have a significantly decreased ability to express several Ad late genes. Additionally, if the E1 gene was present in the infected noncomplementing cells, [**100K**-]Ad vectors were capable of replicating their genomes to high copy number, but were significantly blocked in their ability to efficiently encapsidate the replicated genomes. Injection of an [E1-,**100K**-]Ad vector in vivo also correlated with significantly decreased hepatotoxicity, as well as prolonged vector persistence. In summary, the unique properties of [**100K**-]Ad vectors suggest that they may have utility in a variety of gene therapy applications.

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L4: Entry 3 of 22 File: USPT Dec 10, 2002

DOCUMENT-IDENTIFIER: US 6492343 B1
TITLE: Porcine adenovirus type 3 genome

Other Reference Publication (3):
McCoy et al. Nucleotide and Amino Acid Sequence Analysis of the 100K Protein of a Serotype 3 Porcine Adenovirus. DNA Sequence-The Journal of Sequencing and Mapping, vol. 8, pp. 59-61, 1997.*